Elbow Flexion Contractures in Childhood in Obstetric Brachial Plexus Lesions: A Longitudinal Study of 20 Neurosurgically Reconstructed Infants with 8-Year Follow-up

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Abstract

Objective Little knowledge exists on the development of elbow flexion contractures in children with obstetrical brachial plexus lesion (OBPL). This study aims to evaluate the prognostic significance of several neuromuscular parameters in infants with OBPL regarding the later development of elbow flexion contractures.

Methods Twenty infants with OBPL with insufficient signs of recovery in the first months of life who were neurosurgically reconstructed were included. At a mean age of 4.6 months, the following neuromuscular parameters were assessed: existence of flexion contractures, cross-sectional area (CSA) of upper arm muscles on MRI, Narakas classification, EMG results, and elbow muscle function using the Gilbert score. In childhood at follow-up at mean age of 7.7 years, we measured the amount of flexion contractures and the upper arm peak force (Newton). Statistical analysis is used to assess relations between these parameters.

Results Flexion contractures of greater than 10 degrees occurred in 55% of our patient group. The relation between the parameters in infancy and the flexion contractures in childhood is almost nonexistent. Only the Narakas classification was related to the development of flexion contractures in childhood ($p = 0.006$). Infant muscle CSA is related to childhood peak muscle force.

Conclusion The role of infancy upper arm muscle hypotrophy/hypertrophy, reinnervation, and early elbow muscle function in the development of childhood elbow contractures remains unclear. In this cohort prediction of childhood flexion, contractures were not possible using infancy neuromuscular parameters. We suggest that contractures might be an adaptive process to optimize residual muscle function.

Keywords
- obstetrical brachial plexus lesions
- elbow flexion contractures
- muscle hypotrophy
- muscle atrophy
- MRI
- EMG

Introduction

Obstetrical brachial plexus lesion (OBPL) results in various gradations of paresis or paralysis of the upper limb in infants. In 10 to 30% of the children, OBPL leads to residual deformities of the shoulder, elbow, forearm, or hand.[1–3] Although the development and prevalence of deformities of the shoulder joint in OBPL have been studied extensively,[4,5] there is a lack

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of literature on the development and prevalence of flexion contracture in the elbow joint in these infants.

Contracture formation of the elbow in OBPL has been reported in 48 to 70% of children with OBPL.[6–8] However, up to now, the pathophysiology and origin of elbow flexion contracture in OBPL remain unclear.

Experimental neonatal mice studies[9,10] showed that muscle denervation of the upper arm led to reduced muscle growth (hypotrophy) and resulted in contracture formation of the elbow. Correlation studies in older children with OBPL showed that hypotrophy is related to contractures both in the shoulder and elbow muscle.[4,5,11–14] However, longitudinal studies on the development of contractures of the elbow in children with OBPL are lacking.

This longitudinal prospective study in infants with severe OBPL aims to assess whether muscle growth disturbances, muscle denervation, and muscle imbalance in the upper arm in infancy are related to the development of elbow flexion contractures in childhood.

Methods

Between 1999 and 2006, we prospectively included all newborn infants with unilateral OBPL and insufficient signs of neurological recovery within the first 4 months of life. In all infants the severity of the neural lesion was classified according to Narakas[15] (class I: Deltoid and biceps paresis; C5, C6 lesion; class II: Deltoid and biceps paresis plus paresis of the extensors of the elbow, hand, and fingers, C5, C6, C7 lesion; class III: Almost complete paresis, C5, C6, C7, C8 lesion; class IV: Total arm paresis, Horner syndrome, C5, C6, C7, C8, Th1 lesion).

All infants were clinically assessed on contractures, and muscle function was assessed according to Gilbert and Tassin[16] (►**Table 1**). Magnetic resonance imaging (MRI) was performed to demonstrate the location of the nerve damage and to measure the cross-sectional area (CSA) of the elbow flexors (m. biceps and m. brachialis) and elbow extensors (m. triceps). In addition, electromyographic (EMG) studies were performed to classify the amount of muscle denervation. The unaffected arm was used as a reference to assess the degree of muscle hypotrophy and restriction of movement. MRI was performed under sedation in a standard position with both hands on the abdomen. After visualization of the neural structures (cervical spinal cord and brachial plexus), the imaging continued with visualization of the upper arms using a three-dimensional, fast-imaging, steady-state precession pulse-acquisition sequence imager (repetition time 25 ms, echo time 10 ms, flip angle 40 degrees). The partitions used ranged from 0.8 to 1.5 mm. The protocol included imaging of both the affected and unaffected arms to enable comparison with the unaffected anatomy. MRI measurements of CSA of the elbow flexors (m. biceps and m. brachialis) and elbow extensors (m. triceps) were determined by one of the authors (MSL) in the transverse MRI slide at the most distal humeral insertion of the m. deltoids. CSA of the flexors and extensors was calculated from the MRI using software from Centricity RA 600 (General Electric Health Care, Slough, the United Kingdom) and defined in square millimeters (►**Fig. 1**). Excluded were infants with incomplete MRI of the upper part of the arm or MRI of insufficient quality and infants not neurosurgically reconstructed.

EMG was performed using needle EMG of the biceps and triceps and classified in four categories: (A) denervation, with presence of a solitary pattern, fibrillations, or steep positives waves that implicate spontaneous involuntary muscle action; (B) both denervation and reinnervation, when a mixed pattern is seen; (C) weak reinnervation, concluded if polyphasic potentials seen; and (D) clear reinnervation with (low) average potentials.

All children were treated according to the treatment protocol used for children with OBPL. In all children in this study, surgical plexus reconstruction was indicated and performed according to standard criteria.[17,18]

All children were regularly assessed at the outpatient clinic. Final follow-up was defined at a mean age of 92.8 months (7.8 years, range 4–11 years, SD 23 months). At final follow-up upper arm muscle peak force and assessment of elbow flexion contraction was measured. Also, shoulder passive external rotation in neutral abduction was measured using a goniometer.

Upper arm muscle peak force was measured with the Microfet 2 dynamometer (Biometrics, Almere, the Netherlands) on both the affected and normal side. Measurements were performed by the same orthopaedic surgeon (JAS). During isometric measurement the elbow is held at 90-degree flexion, and the dynamometer is positioned at the wrist. The patients are asked to exercise maximal pressure to the dynamometer. Each measurement is repeated three times, and the median value is used and given in Newton.

The degree of flexion contracture of the elbow is measured with a goniometer after passive positioning of the affected arm in maximum extension.

Table 1 Measurement of muscle function according to Gilbert and Tassin muscle grading score

Score	Clinical description of movement of hand to mouth
M0	No contraction
M1	Contraction, no movement or slight finger movement
M2	Incomplete movement or complete movement without gravity
M3	Complete movement against the weight of the corresponding segment of extremity

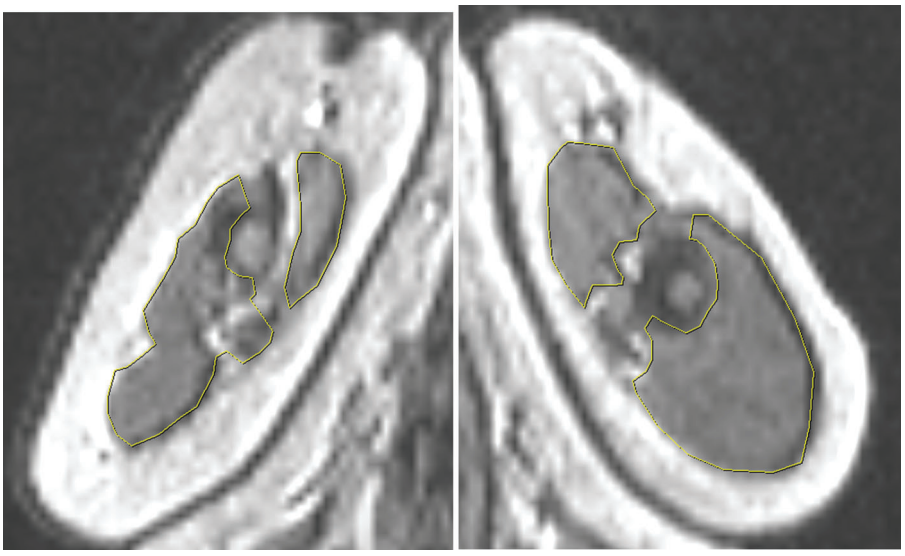

Fig. 1 Cross-sectional area measurement of flexor and extensor muscles in upper arm of 4.5-month-old infant (case 11) with obstetrical brachial plexus lesion. Flexor muscle on upper side of image. Left image: affected arm; right image: unaffected arm.

The study was approved by the Medical Ethical Examination Committee of our institution (nr 2013/274) and informed consent is obtained from the infants' parents.

Statistical Analysis

All data were analyzed using SPSS (version 15.0; SPSS Inc., Chicago, Illinois, United States). Results are presented as means (standard deviation [SD]). To correct for individual differences (like size and age), upper arm force and CSA of the affected side are also expressed as a percentage of the values of the unaffected side. Differences between the affected and unaffected arm were tested, using paired *t*-test for parametric and the Wilcoxon test for nonparametric data; the Pearson's correlation coefficient test for relations between normal distributed variables and the Spearman's correlation coefficient for other distributions. Prediction of contractures was attempted using multiple linear and logistic regressions. All test were two tailed and considered significant if $p < 0.05$.

Results

Twenty children (10 boys) were included with the infancy parameters assessed at the average age of 4.6 months (range 2.1–6.3 months, SD 1 month). The right arm was affected in 8 children, the left in 12. All infants had plexus reconstruction. At the time the measurements were performed in infancy, none of the children had been operated on. An overview of the 20 children characteristics is shown in ►**Tables 2** and **3** and aggregated results are shown in ►**Table 4**.

None of the infants had flexion contractures at the time of inclusion.

Gilbert score of all infants are shown in ►**Table 4**. The mean Gilbert scores of the elbow flexor and extensors were 0.7 and 1.4, respectively. The Gilbert score of the extensors and flexors was not interrelated (Spearman Rho = −0.06, $p = 0.8$).

Initial muscle size CSA measurements on MRI of the flexor and extensor showed both hypotrophy and hypertrophy as compared with the nonaffected side.

In the elbow flexors 15 infants showed hypotrophy (mean 70%, SD 19%) and 5 showed hypertrophy (mean 118%, SD 9%) defined as less than 95% or greater than 105% of the unaffected arm.

In the elbow extensors 13 infants showed hypotrophy (mean 80%, SD 13%) and 7 showed hypertrophy (mean 116%, SD 10%). Pooled data are shown in ►**Table 4**. The CSA of flexors and CSA of the extensors were not interrelated (Spearman Rho = 0.2, $p = 0.3$).

The majority of the infants had signs of weak or clear reinnervation within the biceps or triceps. In the extensors EMG results correlated with the Gilbert score (Spearman Rho = 0.45, $p = 0.02$): Higher reinnervation was related with a higher Gilbert score. In the flexors, no relation with the Gilbert score was found (Spearman Rho = 0.36, $p = 0.1$).

The CSAs of the elbow flexors (m. biceps en m. brachialis) and elbow extensors (m. triceps) were not related to their Gilbert score in infancy nor were their EMG results (Spearman flexor: Rho = 0.02, $p = 0.9$; extensor: Rho = 0.32, $p = 0.17$).

All children were reassessed for final follow-up at a mean age of 92.8 months (7.7 years, range 4–11 years, SD 23 months). Prevalence of flexion contractures of more than 10 degrees was 55%. In these children the mean amount of elbow flexion contractures was 30 degrees (SD 9.2). In the total group flexion contracture was mean 18 degrees (SD 16).

The Narakas classification at infancy was related to the amount of degrees of elbow flexion contractures at childhood (Spearman Rho = −0.6, $p = 0.006$); in Narakas class 3 flexion contractures were more severe than those in Narakas class 1. The Gilbert score was not related to the amount of degrees of flexion contractures in childhood (Spearman test flexors: $p = 0.6$; extensors: $p = 0.9$), nor were CSA measurements

Table 2 Overview of infants characteristics ($n = 20$) and details of neurosurgical reconstruction

10 males, 10 females	
Birth weight (g)	Mean 3,990 (SD 707)
Gestational age	Mean 40 wk (SD 13 d)
OBPL-type Narakas classification	Type 1 ($n = 10$), 2 ($n = 2$), 3 ($n = 8$)
Case	Neurosurgical reconstruction
1	C4 > LD C5 > n. SS, FL; C6 > FL; C7 > TM, FP
2	C5 > FP, TM; C6 > FL, FP;C7 > TM, C8
3	C5 > FP; C6 >FL, FP
4	C5 > FP; C6 >FL, FP; C7 >C5
5	C5 > n. SS;C6 >C5
6	C5 > FP, FL, n SS; C6 >FL, FP
7	C5 >n. SS; C6 >FL; C7 >FP, FL
8	C5 >C5root, FL; C6 >FL, FP
9	C4 >FL, FP; C5 >FP; C6 >FL; TI >TM
10	C5 >FL, FP; C6 >FL, FP
11	C4 >FP, FL, C7; C5 >FP; C6 >FL, C8; C7 >N11
12	C5 >FP, n. SS; C6 >FL, FP
13	C5 >FP ; C6 >FL, C7 >TM
14	C5 >FL, FP; C6 >FL; C7 >C4, TM, FP
15	C5 >FL, FP, n. SS; C6 >FL
16	C5 >PD, LD, n. SS; C6 >LD, PD
17	C5 >FP, FL; C6 >FP, n. SS, C8
18	C5 >T1, FL, FP; C6 >FL, FP; C7 >C8
19	C5 >PD, LD, n. SS; C6 >LD, PD
20	C5 >n. SS, FP, FL ; C6 >FL; C7 >FP, TM

Abbreviations: C4 > , cervical root 4 connected to; FL, fasciculus lateralis; FP, fasciculus posterior; LD, lateral division; n. SS, nervus suprascapularis; OBPL, obstetrical brachial plexus lesion; PD, posterior division; TM, truncus medius; TI, truncus inferior.

and EMG categories related to the amount of elbow flexion contractures in childhood.

The relation between shoulder external rotation and elbow flexion contracture is very weak and nonsignificant (Spearman Rho = 0.1, $p = 0.6$), which suggests that contracture formation is not an infant-related tendency but is dependent on local neuromuscular factors.

At final follow-up upper arm muscle peak-force was significantly weaker on the affected arm, compared with the unaffected arm (Wilcoxon $p < 0.001$, *t*-test) (►**Table 4**). Affected flexors (m. biceps and m. brachialis) peak force was mean 43% (SD = 20%) of the unaffected arm. Affected extensors peak force was mean 60% (SD = 28%) of the unaffected arm.

The upper arm peak force of both flexors and extensors were not related to the amount of flexion contracture (Spearman test extensor: $p = 0.4$ flexor: $p = 0.1$).

The Narakas classification at infancy was related to the upper arm extension peak force (Spearman Rho = −0.44, $p = 0.05$) but not to the flexion peak force (Spearman test $p = 0.7$). A positive relation was found between infant muscle CSA and childhood peak force for extensors (Spearman Rho = 0.6, $p = 0.002$) and for flexors of the upper arm (Spearman Rho = 0.5, $p = 0.01$). The Gilbert score of flexors was related to the upper arm peak force of flexors in childhood (Spearman Rho = 0.4 $p = 0.05$). This relation was not found for the extensors (Spearman Rho = 0.35 $p = 0.12$).

We were not able to create a model predicting elbow flexion contractures, using multivariate techniques (multiple regression and logistic regression) with the infancy parameters as independent variables.

Discussion

Two results emerge from this study. The first is that the Narakas classification in infants with OBPL was related to the development of future elbow flexion contractures in childhood: more contractures in the higher Narakas classes. The second is that, contrary to our hypothesis, we found no relation between the initial measurements of muscle CSA, Gilbert score, and EMG results in infancy and the development of elbow flexion contractures in childhood. Prediction of which infants will develop elbow contractures in childhood was not possible based on infancy parameters used.

In our cohort, the prevalence of elbow flexion contractures was 55%. Because we specifically selected infants with insufficient signs of recovery, our prevalence rate is not comparable with other rates. Possibly, our rate might increase over a period of time since Sheffler et al[7] found an age of onset of elbow contractures up to 14.8 years.

In the present study, no relation was found between hyper/hypotrophy of the elbow flexors (or extensors) in infancy and elbow flexion contractures at final follow-up. This is in contrast to findings in the glenohumeral joint.[5,11,13,14,19,20] Around the shoulder muscle atrophy in infants with OBPL proved to be related to internal rotation contractures and shoulder deformities in childhood. Also, in a correlation study atrophy of the brachialis was related to elbow contracture formation.[12] Unfortunately we could not distinguish the brachialis and biceps hypotrophy in our MRI, but a special role for the brachialis is unexpected in view of the experimental literature.[10] It may be questioned why this relation between hypotrophy and contractures found in the shoulder and confirmed in experimental mice studies of the elbow joint[9,10] was not found in our longitudinal study of the elbow joint.

Possibly muscle size (and thus CSA) changed over the period of time studied: hypotrophic muscles recovered or hypertrophic muscles atrophied. The presence of both hypotrophy and hypertrophy of the muscles in infancy suggests that a range of processes is active in infancy after denervation, which influences CSA and causes the CSA to be variable in size

Table 3 Overview of the cases with OBPL

Case no.	Gender	OBPL side	Age at infancy measurements (mo)	CSA of MRI[a]		Narakas class	EMG results[b]		Gilbert score		Age at follow-up measurements in childhood (mo)	Muscle function[a]		Flexion contracture in degrees
				Flexor	Extensor		Flexor	Extensor	Flexor	Extensor		Flexor	Extensor	
1	F	L	3.1	32	66	1	C	A	0	0	63	25	22	5
2	F	R	5.6	81	78	3	A		0	3	103	11	13	30
3	F	R	4.7	92	100	1	D	D	3	1	92	93	69	20
4	F	R	5.4	132	77	1	D	D	3	0	115	52	38	20
5	F	R	5.1	62	91	1	D	D	0	3	82	22	87	0
6	M	L	5.4	47	78	1	A	D	1	3	111	48	71	10
7	M	L	3.6	117	86	3	A	C	0	0	87	58	64	40
8	M	L	5.0	119	111	1	A	D	0	2	110	28	107	0
9	M	L	4.5	65	80	3		C	0	0	86	36	37	20
10	F	L	5.9	97	88	2	B	B	0	3	74	34	48	30
11	M	R	4.5	82	55	3	D	B	3	0	74	40	2	45
12	F	L	4.7	87	116	1	D	D	2	2	120	40	95	20
13	M	R	6.3	113	138	3	C	C	0	0	69	45	76	30
14	F	L	4.9	81	108	3	D	D	0	3	105	87	80	40
15	M	L	4.1	74	108	2	D	D	0	3	103	29	60	35
16	M	L	4.9	81	108	1	D	D	0	1	45	44	95	10
17	F	L	4.8	86	70	1	D	D	0	2	125	58	70	10
18	M	L	2.1	54	96	3	A	A	0	0	85	32	69	0
19	F	R	5.7	97	120	1	D	D	2	3	136	46	72	0
20	M	R	3.4	108	71	3	D	D	0	0	72	35	32	10

Abbreviations: CSA, cross-sectional area; EMG, electromyography; F, female; L, left; M, male; MRI, magnetic resonance imaging; OBPL, obstetrical brachial plexus lesion; R, right.
[a]As a percentage of the unaffected arm.
[b]A = denervation, B = both denervation and reinnervation, C = weak reinnervation, D = clear reinnervation.

Table 4 Summary of neuromuscular data in infancy and childhood of 20 infants with OBPL

		Unaffected arm, mean (SD)	Affected arm, mean (SD)	Percentage affected/ unaffected, mean (SD)	Mean difference (95% CI of the difference), *p* value
CSA in mm^2	Flexor	240 (42)	198 (51)	85.3% (25)	−41.5 (−74 to −89), $p = 0.015$
	Extensor	412.1 (68)	376 (88)	92.3% (21)	−35.8 (−78 to 6), $p = 0.09$
	Ratio flexor/ extensor	0.59 (0.1)	0.55 (0,16)		0.05 (−0.1 to 0.04), $p = 0.3$
Gilbert score	Flexor	3	0.7 (1.2)		−2.3 (2.9 to 1.8), $p = 0.015$
	Extensor	3	1.4 (1.3)		−1.6 (0.9 to 2.2), $p = 0.000$
Force in Newton	Flexor	79.2 (39)	34.5 (24)	43% (20)	−44.6 (−56.3 to −32.9), $p = 0.000$
	Extensor	56.3 (22)	33.4 (20)	60.3% (28)	−22.9 (−32.4 to −13.4), $p = 0.000$
	Ratio flexor/ extensor	1.3 (0.3)	1.6 (3.8)		0.3 (−1 to 2), $p = 0.7$
Flexion contracture	In degrees	0	17.8 (16)		−17.8 (−25 to −10), $p = 0.000$
	Range of motion in degrees	135	108.5 (15)		26 (20 to 34), $p = 0.000$

Abbreviations: CI, confidence interval; CSA, cross-sectional area; OBPL, obstetrical brachial plexus lesion; SD, standard deviation.

in the first year of life. Yet infant muscle CSA is to some extent relevant, as it is related to childhood peak muscle force both for extensors and flexors.

Narakas classification was related to the development of elbow flexion contracture in childhood. The average degree of elbow flexion contractures is increased in higher Narakas classes. This is partially consistent with the experimental mice study[10] that found that on micropathological level more contractures developed if less than 15% of the axons remain. However, in the same study they also found that isolated upper trunk lesions (C5-C6) and global plexus lesions (C5-Th1) did not differ in the amount of flexion contractures. Whatever the precise relation between number of functioning axons and contractures, other studies also confirm that the number of remaining motor units influence the growth of muscle fibers.[21] The role of denervation was explored by assessing the influence of early EMG findings on contracture development.

Based on the experimental denervation studies, we expected more elbow flexion contractures in children with signs of denervation in the upper arm muscles in infancy, then those with reinnervation. However, this was not confirmed—possibly because of confounding by the neurosurgery since all infants had brachial plexus reconstruction. In an experimental mice study,[10] it was found that immediate brachial plexus reconstruction caused reinnervation and prevented the development of elbow flexion contractures. The brachial plexus reconstruction in our cohort was performed at a mean age of 6 months, and because we always found sequelae, this proved to be beyond the time interval suggested by Weekley et al where it led to complete recovery if indeed such an interval exists in infants. Plexus reconstruction is tailored to the lesion and local anatomy and is rarely standardized. Reinnervation results are uncertain. It is also unpredictable which part of a reconstruction will affect which movement pattern. The C7 root might be dominant in the very young infants; however, with age, its role declines and becomes the least important root. Its role in the prevention of contractures is not clear nor do we know whether adaptations in central motor programs influence muscle development. More knowledge is necessary on the role and interaction between muscle innervation, quality of nerve reconstruction, and the integration of motor-unit patterns. At present we are not able to see the pattern leading to contractures.

As to the relation between residual muscle function and contractures, this is unclear. Several studies have already shown that the intuitively attractive muscle imbalance theory (i.e., that dominance of flexors leads to flexion contractures) cannot explain the development of elbow flexion contractures.[10] On the other hand, Ballinger's and Hoffer's[6] suggestion that flexor weakness correlates with elbow flexion contracture severity is inconsistent with our results. A recent study[22] suggested single muscle overactivity as a factor: overactivity of the long head of the biceps was related to elbow flexion contractures based on EMG findings in older children (mean age 14 years). Our EMG findings are not precise enough to refute or support this hypothesis. The relation between residual muscle function and contractures is unclear.

Various factors have been described that influence the morphology and growth of skeletal muscles in lower limb denervation models.[21,23,24] Nikoloau et al[9] found the brachialis and biceps muscles to shorten by different architectural mechanisms. The biceps showed fibrotic histopathology and sarcomeres of normal length. The brachialis showed fatty infiltration and longer sarcomeres. However, in children reports of pathological findings are inconsistent.[19,25]

Based on our results, we would like to present the hypothesis that elbow flexion contractures are not only scar/recovery mechanisms of the trauma, but they also have an adaptive functional role. The flexion contractures might be a symptom of changes in muscle architecture that aim to optimize the length-force curve of the denervated muscle. Assume that OBPL muscle paresis triggers a remodeling process that improves the remaining myocyte function and leads to a cascade of processes leading to muscle architecture reorganization. This reorganization causes flexion contractures that play a role to optimize the upper arm peak force. This adaptive process is influenced by or dependent on individual differences in brachial plexus anatomy and motor-units organization, differences in plexus lesion, differences in nerve sprouting potential, and differences in primary muscle layout. After the adaptation these differences have confounded any relation between residual muscle function and flexion contracture.

Several limitations are to be noted. A major limitation is that infants had brachial plexus reconstruction after collecting the infancy parameters. The extent, to which surgery affects the denervated muscles in function and size, is unknown. Other individual difference in conservative treatment (physiotherapy, activity level of the child) might influence the results. Another major limitation is that the studied infants represent a limited spectrum of OBPL, as they did not recover enough within 4 months and were being considered for brachial plexus reconstruction. It is unknown to what extent these findings concern the excluded OBPL types.

Other limitations concern the accuracy of the measurements. Differences in elbow flexion during MRI may affect the estimates of muscle CSA. Because the position of the upper arm, with hands placed on the abdomen, is not perpendicular to the transversal magnetic resonance angiography (MRA) scans, the CSA may be slightly overestimated. Small differences in position of both arms during MRI measurements could have affected the CSA. However, calculations in earlier studies[26] show that the effects are minimal. The muscle peak-force measurements are done by a dynamometer which has good validity for isometric peak-force measurements.[27] We realize that isometric muscle peak force depends on muscle length.[28] Standardization of muscle length is attempted by equal positioning, standing with the upper arm against the body with the elbow flexed to 90 degrees, but this does not distinguish in interindividual differences in resting muscle length, as contractures are present in some children. All these may result in peak-force measurement on different lengths in their personal force-length curves. Overuse of the unaffected arm in daily life can influence the peak-force measurement of the unaffected arm. The last limitation is that the classification of the spectrum of EMG findings in the four ordinal classes is debatable; especially in category D (clear reinnervation) the amount of potentials may vary considerably.

Conclusion

In neurosurgically reconstructed OBPL infants, Gilbert score of infancy elbow function, EMG, and MRI-CSA findings of the upper arm muscles are not related to the development of elbow flexion contractures in childhood. Only Narakas classification was related to the development of elbow flexion contracture. Predicting elbow flexion contractures using the infancy parameters was not possible. CSA findings of the upper arm muscles in infants are related to childhood upper arm peak force.

Competing Interest
M.J.S. and J.A.S. are relatives. A competing interest that might influence the results does not exist.

Authors' Contributions
M.J.S.: data acquisition and analysis and writing of the article. W.R.O.: data collection, revising the article. J.A.S.: conception of design, data collection and analysis, drafting manuscript. B.J.R.: writing and structuring of the article.

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